In the Specification:

Please insert the following paragraph on page 1, line 3, after the title and before the "Field of the Invention" heading :

--This application claims the benefit of U.S. Provisional Application Serial No. 60/146,316, filed on July 29, 1999.--

Please replace the paragraph beginning at page 2, line 29 with the following:

--The present invention provides a novel class of oligopeptides that include amino acid sequences containing cleavage sites for human glandular kallikrein (HK2)~~, Fig. 1~~ (see Figure 1). These cleavage sites are derived from an hK2 specific cleavage map of semenogelin I and II~~, Fig. 1~~ (see Figure 1). These oligo-peptides are useful in assays that can determine the free hK2 protease activity. Furthermore, the invention also provides a therapeutic prodrug composition, comprising a therapeutic drug linked to a peptide, which is specifically cleaved by hK2. The linkage substantially inhibits the non-specific toxicity of the drug, and cleavage of the peptide releases the drug, activating it or restoring its non-specific toxicity.--

Please replace the paragraph beginning at page 13, line 9 with the following:

--Thapsigargin is a sesquiterpene-γ-lactone having ~~the following~~ the structure disclosed in International Publication No. WO 98/52966. Primary amines can be placed in substituent groups pendant from either C-2 or C-8 carbon (carbons are numbered as described in International Publication No. WO 98/52966). Preferred primary amine containing thapsigargin analogs that can be coupled to the peptides described above include those described previously by the inventors ("Tissue Specific Prodrug" International Patent Application PCT/US98/10285, published as International Publication No. WO 98/52966, corresponding to USSN 60/047,070

and 60/080,046, filed 5/19/97 and 3/30/98). These primary amine-containing analogs have non-specific toxicity toward cells. This toxicity is measured as the toxicity needed to kill 50% of clonogenic cells (LC_{50}). The ~~LC50~~ LC_{50} of the analogs of this invention is desirably at most 10 μM, preferably at most 2 μM and more preferably at most 200 nM of analog.--

Please replace the paragraph beginning at page 17, line 15 with the following:

--The pH dependence of hK2 was determined using a universal buffer composed of 29 mM citric acid, 29 mM citric acid, 29 mM KH_2PO_4, 29 mM boric acid 0.1 M NaCl and 0.2% bovine serum albumin (BSA). The buffering range is Ph 2.4-11.8. The rate of the cleavage of the substrate I-1295 (100 μM) by 1.6 pmol hK2 was followed for 20 minutes at pH 5, 5.5, 6, 6.5, 7, 7.5, 8, 8.5, 9, 9.5, and 10.--

Please replace the paragraph beginning at page 21, line 6 with the following:

--The inhibition of hK2 by Zn^{2+} was studied using the substrates ~~Pro-Phe-arg-AMC~~ Pro-Phe-Arg-AMC (SEQ ID NO:55) and Ala-Arg-Arg-AMC (SEQ ID NO:62) at concentrations varying from 9 to 180 μM and $ZcCl_2$ concentrations ranging from 0.5 μM to 1mM. Since BSA contains several binding sites for Zn^{2+} it could not be used in the kinetics buffer during the zinc inhibition experiments. The binding of hK2 to the microtiter well walls caused a constant decrease in the reaction rate, which was however similar to all zinc concentrations. The velocities were calculated from a five-minute measurement time after mixing of the enzyme with the buffer containing substrate and zinc.--

Please replace the paragraph beginning at page 21, line 13 with the following:

--The enzymatic activity of hK2 was inhibited by zinc ions at micromolar concentrations, and the inhibition was totally reversed by addition of EDTA. The inhibition of hK2 by zinc was first tested against competitive, uncompetitive, mixed, non-competitive, and partial non-competitive inhibitor models using commonly used formulas described for the respective inhibition models. Zn^{2+} both increased the K_m and decreased the V_{max}. The Dixon plots ~~1/v versus [Zn^{2+}]~~ [Zn^{2+}]/v) for the inhibition were not linear. However, at low zinc ion concentrations the inhibition pattern looked competitive. The inhibition mechanism is clearly more complex than the ones described by the formulas used. Further analysis of the inhibition mechanism was done by deriving the rate equations for various more complex mechanisms and analyzing the data by least-squares best-fit systems. The possible mechanism required two bound zinc ions, and is presented by Scheme 1. In the best mechanism, the first bound zinc ion does not cause inhibition (k = k', or k' was even slightly higher than k).--